Report SAM-TR-78-17

ABSENCE OF HIGH-G STRESS CARDIOPATHY IN A HUMAN CENTRIFUGE RIDER

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May 1978

Final Report for Period April 1975 - April 1978

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NOTICES

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SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

REPORT DOCUMENTATION PAGE

READ INSTRUCTIONS BEFORE COMPLETING FORM

1. REPORT NUMBER: 14 SAM-TR-78-17
2. GOVT ACCESSION NO.:
3. RECIPIENT'S CATALOG NUMBER:
4. TITLE (and Subtitle): 6 ABSENCE OF HIGH-G STRESS CARDIOPATHY IN A HUMAN CENTRIFUGE RIDER.
5. TYPE OF REPORT & PERIOD COVERED: 9 Final rept. Apr [redacted]75 - Apr [redacted]78,
6. PERFORMING ORG. REPORT NUMBER:
7. AUTHOR(s): 10 Kent K. Gillingham M.D., Ph.D.
8. CONTRACT OR GRANT NUMBER(s):
9. PERFORMING ORGANIZATION NAME AND ADDRESS: USAF School of Aerospace Medicine (VNB) Aerospace Medical Division (AFSC) Brooks Air Force Base, Texas 78235
10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS: 62202F 16 7930-12-20 17 12
11. CONTROLLING OFFICE NAME AND ADDRESS: USAF School of Aerospace Medicine (VNB) Aerospace Medical Division (AFSC) Brooks Air Force Base, Texas 78235
12. REPORT DATE: 11 May [redacted]78
13. NUMBER OF PAGES: 10
14. MONITORING AGENCY NAME & ADDRESS (if different from Controlling Office): 12 12p.
15. SECURITY CLASS. (of this report): UNCLASSIFIED

15a. DECLASSIFICATION/DOWNGRADING SCHEDULE

16. DISTRIBUTION STATEMENT (of this Report)

Approved for public release; distribution unlimited.

17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)

18. SUPPLEMENTARY NOTES

19. KEY WORDS (Continue on reverse side if necessary and identify by block number)

Acceleration
G stress
Stress cardiopathy
Cardiomyopathy

20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

After a USAFSAM Acceleration Stress Panel member was fatally injured in a traffic accident, his heart was examined for evidence of high-G stress cardiopathy. None was found. An hypothesis relating high-G stress cardiopathy in experimental animals to cardiotoxicity of catecholamines liberated by the psychic stress of centrifugation is presented.

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UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

317000

PREFACE

The enthusiastic and invaluable assistance of Lt Col William F. MacKenzie, USAF, VC, USAFSAM/VSP, in the pathologic examination of Airman X's heart is sincerely appreciated. Dennis D. Reichenbach, M.D., of the University of Washington School of Medicine, generously donated his time and resources in providing the electron microscopic examination of the tissue, after serving as prime consultant in the light microscopic examination. Col Robert McMeekin, USA, MC, of the Aviation Pathology Branch, AFIP, also provided valuable assistance in the pathologic examination. Special thanks are extended to Col Grant B. McNaughton, USAF, MC, Aerospace Medicine Section, Edwards AFB, CA, for his resourceful and persevering cooperation in obtaining the tissue specimens for examination. Most importantly, we owe a profound debt of gratitude to Airman X's parents for their remarkable understanding and cooperation in this effort during the emotionally trying period following the tragic loss of their son.

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ABSENCE OF HIGH-G STRESS CARDIOPATHY IN A HUMAN CENTRIFUGE RIDER

INTRODUCTION

For the past six years we have been concerned that sustained high-G stress (>+6 G_z for 15 s or more) might cause heart damage in pilots of F-15, F-16, and other highly maneuverable fighter aircraft. Studies of miniature swine exposed acutely to G stress on the order of +9 G_z for 45 s have consistently revealed subendocardial hemorrhage (3, 4) and cardiomyopathy (12, 13), the latter consisting mainly of myofibrillar degeneration, translocation and clumping of mitochondria, and actual necrosis of cardiac myocytes. Myocardial biochemical systems of similarly G-stressed swine have also been shown to suffer alterations suggestive of acute damage and subsequent repair (5). Interestingly, cardiac function of invasively instrumented swine exposed acutely to +9 G_z for 60 s was not diminished (1), nor was noninvasively assessed cardiac function detectably diminished in human centrifuge riders exposed to 8- and 10-G loads for various durations and at various seatback angles (6). Attempts to detect rises in human cardiac isoenzymes after high-G stress have been consistently unsuccessful (6, 16, 17). Repeated exposures of miniature swine to high +G_z stress tend not to elicit repeated bouts of subendocardial hemorrhage (2, 3), although repeated--possibly *cumulative--cardiomyopathy may* occur (2). Moreover, swine exposed acutely to the procedure of centrifugation, but without actually incurring high-G stress, also suffer subendocardial hemorrhage and cardiomyopathy (2). The pathologic changes seen in hearts of swine exposed to G stress (or to the prospect of G stress) are likewise found in at least some swine succumbing to the so-called porcine stress syndrome (9); this affliction of swine being transported to slaughter is manifested by acute cardiac failure, with cardiotoxic (8, 15) levels of catecholamines--liberated by mental stress--recently having been implicated as causal (10).

A working hypothesis based on the foregoing facts is that the novelty of the centrifugation experience provokes such powerful sympathetic responses in swine that cardiopathic changes occur. The inference is that fighter pilots and human centrifuge riders, although exposed to high-G stress during real and simulated aerial combat maneuvering (ACM), do not suffer cardiopathic changes from the exposures, since they do not experience the extreme psychic stress experienced by the animals. Indirect evidence that such is the case is the fact that heart rates of centrifuged swine commonly reach 270 (mean, 246) beats/min during their first exposures to G stress (2), but rarely do human centrifuge riders' heart rates exceed 190 beats/min during G stress (author's observations), even though resting heart rates of swine and humans are similar. In fact, the mean maximum heart

rate of well-trained human centrifuge riders exposed to a sustained $+9\ G_z$ for 45 s was only 167 beats/min (14); this relatively low rate was in turn higher than the means of 149, recorded from fighter pilots during vigorous ACM in the F-4 (11), and 138, recorded from test pilots pulling 7 G in the YF-16 (7). Such physiologic heart rate responses argue against sympathetic stress-response cardiopathy occurring in humans exposed to high-G stress, but the absence of human pathologic data has heretofore rendered this argument ineffectual. The unfortunate and untimely death of a USAFSAM Acceleration Stress Panel member has, however, provided certain relevant pathologic data which will be related in this report.

CLINICAL HISTORY

Airman X underwent a Flying Class II physical examination on 19 April 1975, at age 19, prior to his being placed on orders allowing him to participate in hazardous duty on the USAFSAM Human Centrifuge. Height was 193 cm; weight, 88.5 kg; BP, 118/78 sitting. The only abnormality noted, other than mild myopia corrected by glasses, was a well-healed, 5-cm scar on his left palm, the result of an injury incurred five years earlier. Airman X began riding the centrifuge on 7 July 1975, and rode consistently for the following 20 months, averaging 2.3 days of G-stress exposure per month. During this time the subject had only one documented episode of ill health, an upper respiratory infection associated with pharyngitis and possible epiglottitis beginning in August 1976 and continuing for several months. No evidence of injury related to G stress was ever revealed, either by symptoms or by postrun physical examinations, with the exception of the cutaneous petechiasis over the arms, legs, and posterior axillary folds, commonly seen after sustained high-G stress (6, 18). The only other significant medical history is that of a left radial arterial cannulation and cannulation of a left dorsal wrist vein on separate occasions in conjunction with high-G experimentation: no complications resulted.

PATHOLOGIC FINDINGS

On 20 February 1977, Airman X was killed in a motorcycle accident. Death was instantaneous, a result of severe injuries to the head. Because of our particular interest in heart pathology associated with G stress, we retained the heart for detailed examination. The heart weighed 360 g and had normally distributed coronary vessels free of atheromata. The endocardial surface was completely normal, with no evidence of either recent or previous subendocardial hemorrhage. A 3- x 3-cm portion of the posterior epicardium which was darker than the remaining myocardium was judged to be livor mortis. Neither the natural surfaces nor the cut surfaces of the myocardium showed fibrotic changes. All valves, chordae tendineae, and papillary muscles were normal. Sections of myocardial tissue taken from the basal left ventricular

septum and from the left anterior papillary muscle, sites of predilection for subendocardial hemorrhage and cardiomyopathy in G-stressed swine, were examined under light microscopy both for acute myopathic changes and for fibrosis. Only rare, isolated myocytes with contraction bands were identified, and these pathologic changes were definitely of agonal origin. No necrosis of myocytes was seen, nor was there evidence of fibrous replacement of myocytes. Further examination by electron microscopy was accomplished, but yielded no additional useful information.

G-STRESS HISTORY

Fortunately, a fairly accurate quantification of Airman X's G-stress exposure on the Human Centrifuge was possible because the type, magnitude, and duration of the acceleration profiles to which he was subjected were documented in his records. Listed in Table 1 are five parameters which characterize his exposure.

TABLE 1. G-STRESS HISTORY OF USAFSAM ACCELERATION STRESS PANEL MEMBER

Month	Days of exposure	Number of exposures	Peak G	G-seconds above 1 G	G-seconds above 4 G
Jul 75	3	16	7	823	187
Aug	4	17	7	2,678	829
Sep	1	2	7	750	255
Oct	1	3	4	96	0
Nov	2	25	7	2,237	428
Dec	1	2	3	53	0
Jan 76	3	18	8	7,150	1,975
Feb	1	6	9	2,530	805
Mar	3	18	10	2,702	688
Apr	1	1	3	30	0
May	1	1	3	30	0
Jun	0	0	1	0	0
Jul	2	11	7	1,450	375
Aug	2	6	7	1,478	445
Sep	1	3	7	417	120
Oct	3	6	7	1,335	313
Nov	3	17	7	2,822	971
Dec	1	1	3	30	0
Jan 77	8	24	7	4,209	959
Feb	5	15	7	3,039	773
Sum	46	192	--	33,859	9,123
Mean	2.30	9.60	--	1,693	456
S.E.	0.41	1.86	--	404	112

The number of days of G-stress exposure per month, the number of separate exposures (runs) per month, and the peak G level attained in a given month are self-explanatory; G-seconds is simply the integral of G load (above 1 G) over time spent riding the centrifuge in a given month; similarly, G-seconds above 4 G is the monthly integral of G load in excess of 4 G over time spent above 4 G. Graphic representations of G-seconds and G-seconds above 4 G are provided in Figure 1.

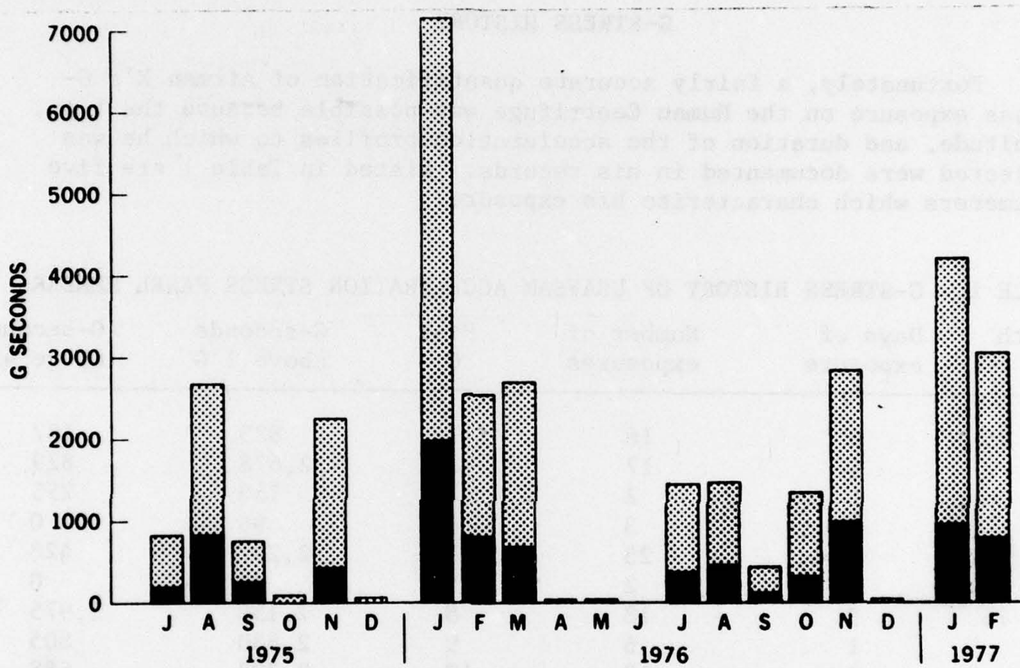

Figure 1. G-stress exposure of Airman X, July 1975 through February 1977. Bars represent monthly integral of G load (>1.0 G) over time; black portions represent integral of G load above 4.0 G.

Although the data indicate substantially greater G-stress exposure during the first three months of 1976 than at other times, almost all of that exposure was effectively at a 65° seatback angle: the $+G_z$ component of the total G stress during those months is approximated by multiplying the total by cos 65°, or 0.42. The G-stress profiles ridden during those three months were like that shown in Figure 2, with a 4 G plateau and three 10-s peaks to 6, 7, 8, 9, or 10 G.

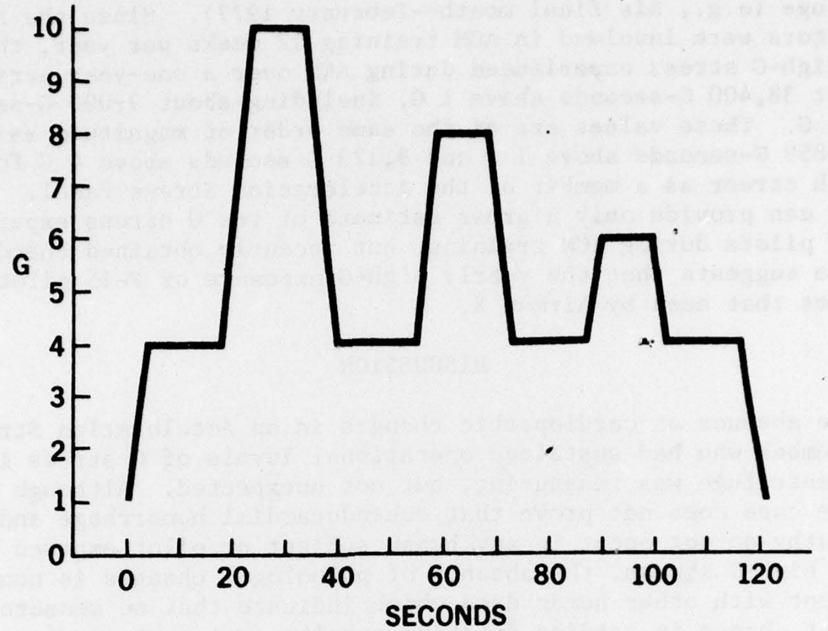

Figure 2. Typical G-stress profile experienced by Airman X on USAFSAM Human Centrifuge. This particular profile was used to evaluate air-bag slant-seat for High Acceleration Cockpit program during first three months of 1976.

During the latter half of his career as a centrifuge rider, Airman X was heavily engaged in development and testing of an improved anti-G valve, and was exposed exclusively to $+G_z$ stress, frequently to a profile consisting of a 4.5-G plateau with up to four 7-G, 15-s peaks. His last high-G exposure was to such a profile, two days before his death.

How does the G-stress history of Airman X compare with that of the average fighter pilot, or with that expected to be experienced by F-15 and F-16 pilots? Leverett et al. (11, and personal communication) measured the G stress sustained by F-4E instructor pilots during ACM training in the Fighter Weapons Instructor Course at the Tactical Fighter Weapons School, Nellis AFB, Nevada, in 1971. One more or less typical aerial combat engagement lasted 100 s, twice peaked briefly (<5 s) at 8 G in a varying G-stress profile which integrated to 320 G-seconds above 1 G and 75 G-seconds above 4 G. Ten such engagements were accomplished in a typical week, so the integrated G stress experienced by these instructor pilots in one week of ACM training--3200 G-seconds above 1 G, 750 G-seconds above 4 G--was roughly equivalent to

what Airman X experienced in a particularly busy month on the Human Centrifuge (e.g., his final month--February 1977). Since the Nellis instructors were involved in ACM training 12 weeks per year, their total high-G stress experienced during ACM over a one-year period would be about 38,400 G-seconds above 1 G, including about 9,000 G-seconds above 4 G. These values are of the same order of magnitude as Airman X's 33,859 G-seconds above 1 G and 9,123 G-seconds above 4 G for his 20-month career as a member of the Acceleration Stress Panel. At this time we can provide only a gross estimate of the G stress experienced by F-15 pilots during ACM training, but recently obtained anecdotal evidence suggests that the yearly high-G exposure of F-15 pilots may be ten times that seen by Airman X.

DISCUSSION

The absence of cardiopathic changes in an Acceleration Stress Panel member who had sustained operational levels of G stress in the Human Centrifuge was reassuring, but not unexpected. Although this one negative case does not prove that subendocardial hemorrhage and cardiomyopathy do not occur in any human subject or pilot exposed repeatedly to high-G stress, the absence of pathologic changes is nonetheless consistent with other human data which indicate that no measurable permanent change in cardiac function results from acute exposure to G stress in the 7- to 10-G range (6). Whether pilots of the F-15, F-16, and other upcoming fighter aircraft will be able to engage in maximum-performance ACM training regularly and repeatedly without harm is another matter, however, since the cumulative G stress associated with such flying is likely to be an order of magnitude greater than that to which Airman X was exposed on the Human Centrifuge. We expect these pilots to suffer no temporary or permanent heart damage from such stress so long as their heart rates remain at physiologic levels (i.e., below 200 beats/min). This expectation rests on the assumption that catecholamine-induced cardiopathy, such as seen in the porcine stress syndrome, would be unlikely under conditions wherein excessive heart rates are not provoked. It would seem reasonable, however, to record heart rates of F-15/F-16 pilots during their most vigorous ACM training missions, and to assess circulating catecholamine levels in trained experimental subjects during centrifuge simulations of these missions. In this manner, needed indirect evidence could be obtained concerning whether current operational G-stress levels are likely to result in significant cardiac strain.

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